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NEWS	2		"Ask CAS" for self-help around the clock
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NEWS	4	May 12	Polymer links for the POLYLINK command completed in REGISTRY
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NEWS EXPRESS	JULY 30 CURRENT WINDOWS VERSION IS V7.01, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
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=> s cardiomyopathy
L1 62357 CARDIOMYOPATHY

=> s heart attack or myfarction
L2 2966 HEART ATTACK OR MYFARCTION

=> s l1 and l2
L3 19 L1 AND L2

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 13 DUP REM L3 (6 DUPLICATES REMOVED)

=> d 1-13 ab,bib

L4 ANSWER 1 OF 13 CA COPYRIGHT 2004 ACS on STN DUPLICATE 1
AB A review. Atherosclerosis is the major cause of cardiovascular disease. Hypercholesterolemia, hypertension and cigarette smoking are the common risk factors for atherosclerosis. These risk factors unite behind a convergence of mechanism, involving oxidation and inflammation in the artery wall that, with time, gives rise to characteristic fatty-fibrous lesions. Phys. trauma and inflammation produce lesion rupture, which can lead to clin. events such as **heart attack** and stroke, or resolve with plaque growth. Disease progression is marked by the inflammatory indicator CRP (C-reactive protein). Early indicators of **heart attack** are the inflammatory marker CD40, and the cardiac myofilament protein troponin. Coronary atherosclerosis is the common cause of heart failure (HF). Disordered calcium signaling to the myofilaments occurs in HF and in **cardiomyopathy**. Enhanced calcium signaling suppresses HF. Neuro-humoral and biomech. processes, as seen in hypertension, produce cardiac hypertrophy, which predisposes to HF through apoptosis. Although in humans cardiac damage produces permanent loss of cells, because the heart cannot regenerate, developments in stem cell technol. suggest that help is at hand.
AN 141:86459 CA
TI Pathophysiology and biochemistry of cardiovascular disease
AU Scott, James
CS Imperial College London, London, SW7 2AZ, UK
SO Current Opinion in Genetics & Development (2004), 14(3), 271-279
CODEN: COGDET; ISSN: 0959-437X
PB Elsevier Science Ltd.
DT Journal; General Review
LA English
RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 13 CA COPYRIGHT 2004 ACS on STN DUPLICATE 2
AB A review. Emotional or phys. stress triggers "tako-tsubo" **cardiomyopathy** or "transient left ventricular apical ballooning", but the pathogenesis is unclear. In response to the immobilization stress

of rats, a useful model of emotional stress, rapid activation of p44/p42 mitogen-activated protein kinase was observed in the heart, followed by a transient upregulation of immediate early genes in the smooth muscle cells of coronary arteries, the endothelial cells and the myocardium. Heat shock protein 70 was induced in the aortic and coronary arterial smooth muscle cells and in the myocardium. Natriuretic peptide genes were also upregulated in the myocardium. Sequential gene expression can be considered as an adaptive response to emotional stress. Blocking of both α-adrenoceptors and β-adrenoceptors eliminated the upregulation of immediate early genes induced by stress, while α-agonists and β-agonists upregulated immediate early genes in the perfused heart. Activation of α-adrenoceptors and β-adrenoceptors is the primary trigger of emotional stress-induced mol. changes in the heart.

AN 138:185005 CA

TI Molecular mechanism of emotional stress-induced and catecholamine-induced **heart attack**

AU Ueyama, Takashi; Senba, Emiko; Kasamatsu, Ken; Hano, Takuzo; Yamamoto, Katsuhiro; Nishio, Ichiro; Tsuruo, Yoshihiro; Yoshida, Ken-ichi

CS Department of Anatomy and Cell Biology, Wakayama Medical University, Wakayama, 641-8509, Japan

SO Journal of Cardiovascular Pharmacology (2003), 41(Suppl. 1), S115-S118
CODEN: JCPCDT; ISSN: 0160-2446

PB Lippincott Williams & Wilkins

DT Journal; General Review

LA English

RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 13 CA COPYRIGHT 2004 ACS on STN

AB New peptides, e.g. Ptu1, Ado1, and Iob1, from venomous saliva of assassin bugs (Peirates turpis, Agriosphodrus dohrni, and Isyndus obscurus) are claimed as N-type calcium channel blockers for treatment of calcium channel-related diseases, including hypertension, **heart attack**, **cardiomyopathy**, arrhythmia, cerebral ischemia, and other cardiovascular diseases. Formulation examples of injections were given.

AN 136:257249 CA

TI New peptides from venomous saliva of assassin bugs as calcium channel blockers

IN Nakashima, Terumi; Korzo, Gerald; Nagao, Hiroshi; Akabane, Satomi

PA Suntory, Ltd., Japan

SO Jpn. Kokai Tokkyo Koho, 26 pp.
CODEN: JKXXAF

DT Patent

LA Japanese

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 2002080499	A2	20020319	JP 2000-266187	20000901
PRAI	JP 2000-266187		20000901		

L4 ANSWER 4 OF 13 CA COPYRIGHT 2004 ACS on STN DUPLICATE 3

AB A review. Fabry disease (FD, OMIM 301500) is an X-linked inherited disorder of metabolism due to mutations in the gene encoding α-galactosidase A, a lysosomal enzyme. The enzymic defect leads to the accumulation of neutral glycosphingolipids throughout the body, particularly within endothelial cells. Resulting narrowing and tortuosity of small blood vessels lead to tissue ischemia and infarction. Inability to prevent the progression of glycosphingolipid deposition causes significant morbidity (acroparesthesia, angiokeratoma, autonomic dysfunction, **cardiomyopathy** and deafness), and mortality from early onset strokes, **heart attack** and renal failure in adulthood. Demonstration of α-galactosidase A deficiency in leukocytes or plasma is the definitive method for the diagnosis of

affected hemizygous males. Most heterozygotes present with a cardiac, renal or neurol. symptomatol., although to a lesser extent than what is observed in hemizygotes. Due to random X-chromosomal inactivation, enzymic detection of carriers is often inconclusive. Mol. testing of possible carriers is therefore mandatory for accurate genetic counseling. The GLA gene has been cloned and more than 200 mutations have been identified. Medical management is symptomatic and consists of partial pain relief with analgesic drugs (gabapentin, carbamazepine), whereas renal transplantation or dialysis is available for patients experiencing end-stage renal failure. However, the ability to produce high doses of α-galactosidase A in vitro has opened the way to clin. studies and enzyme replacement therapy has recently been validated as a therapeutic agent for FD patients in clin. trials. Long term safety and efficacy of replacement therapy are currently being investigated.

AN 138:151023 CA
TI Fabry disease (α-galactosidase A deficiency): pathophysiology, clinical signs and genetics aspects
AU Germain, Dominique Paul
CS Unite de Genetique Clinique, Hopital Europeen Georges Pompidou, Paris, 75015, Fr.
SO Journal de la Societe de Biologie (2002), 196(2), 161-173
CODEN: JDSBFG; ISSN: 1295-0661
PB Masson Editeur
DT Journal; General Review
LA French
RE.CNT 88 THERE ARE 88 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 13 MEDLINE on STN
AB Your patient is in distress, and all an ECG can tell you is that he has had a previous **heart attack**. When are 2-dimensional (2-D) echocardiographic and Doppler studies appropriate options, and what special information can they provide? In the case presented, the Doppler recording profiled the hemodynamic status of the patient. Although the 2-D echocardiogram provided valuable information, only the Doppler study is shown to illustrate how sophisticated hemodynamic information can be gathered from Doppler examination. Check your review of the recording with the discussion on the next page.
AN 2002679015 MEDLINE
DN PubMed ID: 12439351
TI Doppler hemodynamics.
AU Pandian Natesa G
CS Tufts-New England Medical Center, Boston, Massachusetts, USA.
SO Reviews in cardiovascular medicine, (2002 Winter) 3 (1) 57-9.
Journal code: 100960007. ISSN: 1530-6550.
CY United States
DT (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200302
ED Entered STN: 20021120
Last Updated on STN: 20030225
Entered Medline: 20030224

L4 ANSWER 6 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:380805 BIOSIS
DN PREV199800380805
TI Can disease models be replaced by in vitro methods?.
AU Eschenhagen, Thomas [Reprint author]
CS Univ.-Krankenhaus Eppendorf, Pharmakol. Inst., Hamburg, Germany
SO Arzneimittel-Forschung, (March, 1998) Vol. 48, No. 3, pp. 337-338. print.
Meeting Info.: Symposium of the Paul Martini Foundation (Disease Models in Drug Research). Mainz, Germany. November 28-29, 1997. Paul Martini

Foundation.
CODEN: ARZNAD. ISSN: 0004-4172.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA German
ED Entered STN: 2 Sep 1998
Last Updated on STN: 2 Sep 1998

L4 ANSWER 7 OF 13 MEDLINE on STN
AB Patients with apical hypertrophy have a natural favourable history. Non Specific Ventricular Tachycardia (NSVT) at ambulatory monitoring is more favourable if it is not associated with consciousness disorders. A high rate of NSVT episodes at Holter or the association with syncope can lead to a less favourable prognosis and therefore require pharmacological or electric treatment. The role of ET (electrophysiological test) has not yet been clearly described and is in progress. Recent studies of molecular genetics help to identify high-risk patients. Sustained monomorphic VT is not frequent but when it occurs it should be treated with BT. Patients with a light risk of VT should be treated with pharmacological therapy (white amiodarone and/or sotalol) and preferably with implantable defibrillator (ID) if VT cannot be eliminated. ID should be implanted also in the few patients surviving **heart attack** to avoid the risk of relapses.
AN 1998150577 MEDLINE
DN PubMed ID: 9489319
TI [Ventricular arrhythmia in hypertrophic **cardiomyopathy**. When and how to treat].
Le aritmie ventricolari nella miocardiopatia ipertrofica. Quando e come trattarle.
AU Tullio D; Valerio A; Tucci C
CS Servizio di Cardiologia ed UTIC, ULSS Lanciano, Vasto Ospedale Renzetti, Lanciano, Chieti.
SO Minerva cardioangiologica, (1997 Oct) 45 (10) 495-501.
Journal code: 0400725. ISSN: 0026-4725.
CY Italy
DT Journal; Article; (JOURNAL ARTICLE)
LA Italian
FS Priority Journals
EM 199803
ED Entered STN: 19980407
Last Updated on STN: 19980407
Entered Medline: 19980325

L4 ANSWER 8 OF 13 CA COPYRIGHT 2004 ACS on STN
AB Coenzyme Q-10 (CoQ-10) is a vitamin-like, naturally-occurring compound found in most cells of the body as well as in many foods. The mol. structure and the 3-dimensional structure of coenzyme Q-10 are given. This compound plays a key enzymic role in energy production within mitochondria, specialized organelles that function as energetic powerplants to produce ATP (ATP), the metabolic coin-of-the-realm for both plant and animal cells. CoQ-10 has been on the market in Japan since the early 1970s, where it is used as a tonic by approx. 10% of the adult population. For the last decade or so American cardiologists have been using CoQ-10 as a prescription drug (under the trade name Ubiquinone, supplied as 25, 60, or 200 mg tablets by Vitaline Formulas of Ashland, OR) for patients suffering from heart disease. (Other related names are Idebenone, Avan, and CV-2619).1. However, since the mid-1980s, CoQ-10 has been marketed as a nutrient in U.S. health food stores in capsules that range from 10 to 30 mg. Numerous clin. studies have found evidence that CoQ-10 is effective in treating heart failure from primary **cardiomyopathy**, angina (heart pain), certain consequences of myocardial infarction (**heart attack**), and possibly other cardiovascular diseases. There are preliminary reports that CoQ-10 may have anticancer properties. Although its mechanism of action is currently unknown, CoQ-10 is speculated to

function as certain other vitamins do, as an antioxidant. Expts. with rodents have shown that CoQ-10 confers significant advantage in increasing average, but not maximum, life expectancy. We are disappointed to report that coenzyme Q-10 does not appear to increase maximum life span.

AN 126:182887 CA
TI Coenzyme Q-10 and lifespan extension
AU Coles, L. Stephen; Harris, Steven B.
CS Jet Propulsion Laboratory, California Institute of Technology, Pasadena, CA, USA
SO Advances in Anti-Aging Medicine (1996), 205-215. Editor(s): Klatz, Ronald M. Publisher: Liebert, Larchmont, N. Y.
CODEN: 64BDAG
DT Conference
LA English

L4 ANSWER 9 OF 13 CA COPYRIGHT 2004 ACS on STN DUPLICATE 4
AB A review with 50 refs. The mammalian heart is normally well-oxygenated and anaerobic glycolysis is extremely rare except for the production of extra ATP during extreme exercise like a marathon race. Anaerobic glycolysis plays a role when there is a serious impairment in coronary blood flow such as during **heart attack** and open heart surgery.
The control of glycolysis in ischemic myocardial tissue appears to be extremely complex. During aerobic glycolysis, phosphofructokinase is the most important regulatory enzyme that controls the energy requirements of the cell. Under anaerobic conditions, however, glyceraldehyde 3-phosphate dehydrogenase becomes the key enzyme because it responds promptly to any changes in the essential supply of co-factors for oxidation The conversion of pyruvate to acetyl CoA (aerobic metabolism) involves a series of chain reactions primarily catalyzed by pyruvate dehydrogenase complex which is situated at the cross roads between both aerobic and anaerobic glycolysis. It is important to remember that substrate utilization is carefully controlled by substrate availability. During aerobic metabolism, control mechanisms using fatty acids, lactate and glucose as energy substrates regulate the rate of ATP production according to energy demand. This precise mechanism is upset during ischemia and post-ischemic reperfusion for reasons discussed in this review. The demand for ATP can no longer be met by its supply because of severely reduced anaerobic glycolysis and significantly inhibited β-oxidation of fatty acids. The impairment of bioenergetics is discussed in the context of several diseases such as **cardiomyopathy**, heart failure, diabetes, arrhythmias, cardiac surgery, heart-lung transplantation, and also in aging and oxidative stress. The regulation of energy metabolism in preconditioned heart is also discussed. Finally, methods used to preserve energy in ischemic myocardium are summarized and the quantitation of the high-energy phosphates is discussed. This review challenges scientists to discover drugs which will stimulate energy supply during myocardial ischemia.

AN 125:298152 CA
TI Bioenergetics, ischemic contracture and reperfusion injury
AU Das, D. K.; Maulik, N.
CS Dep. of Surgery, Univ. of Connecticut Sch. of Medicine, Farmington, CT, 06030-1110, USA
SO EXS (1996), 76(Myocardial Ischemia: Mechanisms, Reperfusion, Protection), 155-173
CODEN: EXSEE7; ISSN: 1023-294X
PB Birkhaeuser
DT Journal; General Review
LA English

L4 ANSWER 10 OF 13 MEDLINE on STN
AB A 4 1/2 months old female baby was admitted to our hospital after an unexpected **heart attack**. Birth was in the 37th gestational week after an uneventful pregnancy and delivery by sectio, birth weight 1650 g, Apgar 9/10/10. In the following weeks the baby showed general muscle hypotonia, failure to thrive and sometimes an

uncharacteristic heart murmur. Besides a chronic lactic acidemia we found a hypertrophic **cardiomyopathy**, cataract and small defects of the pigment epithelium of the retina. The CT-scan of the brain showed hypodense areas of both thalami and the mid-brain. Metabolic examination of two muscle specimens showed a deficiency of cytochrome-c-oxidase activity (I: 30, II: 20, normal: 73-284 mU/mg protein). So our patient may be the first case with an established defect in the respiratory chain suffering from **cardiomyopathy**, cataract and mitochondrial dysfunction. There is also a strong similarity to other encephalomyopathies especially to the Leigh-Syndrome.

AN 89038146 MEDLINE
DN PubMed ID: 2846943
TI [Encephalomyelopathy, **cardiomyopathy**, cataract and changes in the retinal pigment epithelium resulting from a cytochrome c oxidase deficiency].
Encephalomyelopathie, Kardiomyopathie, Kataract und Pigmentepithelveranderungen der Retina infolge eines Cytochrom-c-Oxidase-Mangels.
AU Sieverding L; Schmaltz A A; Apitz J; Sengers C A; Ruitenbeek W; Trijbels J M; Schroth G
CS Abteilung fur padiatrische Kardiologie, Universitatskinderklinik Tubingen.
SO Klinische Padiatrie, (1988 Sep-Oct) 200 (5) 381-7. Ref: 59
Journal code: 0326144. ISSN: 0300-8630.
CY GERMANY, WEST: Germany, Federal Republic of
DT (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW OF REPORTED CASES)
LA German
FS Priority Journals
EM 198812
ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19881201

L4 ANSWER 11 OF 13 MEDLINE on STN
AB Thirty-six cases of **heart attack** or sudden death in marathon runners have been reported in the world literature to date. The mean age of the runners was 43.8 yr (range = 18 to 70), the mean years' running was 6.8 yr (range = 0.5 to 29), and the mean best standard 42.2 km marathon time was 3 h 28 min (range = 2 h 33 min to 4 h 28 min). Coronary artery disease was diagnosed either clinically, angiographically, or at autopsy in 27 runners (75%), two of whom also had histological evidence of hypertrophic **cardiomyopathy**. Seventy-one percent of the runners with coronary artery disease had premonitory symptoms, and most ignored such symptoms and continued to train or race. Fifty percent of all cardiac events occurred either during or within 24 h of competitive running events or long training runs. The marathon running population does not constitute solely persons with excellent cardiovascular health. Marathon runners, especially those with a family history of heart disease and other coronary risk factors, should not consider themselves immune to either sudden death or to coronary heart disease and should seek medical advice immediately if they develop any symptoms suggestive of ischemic heart disease. Physicians should not assume that "physically fit" marathon runners cannot have serious, life-threatening cardiac disease.
AN 87256889 MEDLINE
DN PubMed ID: 3298928
TI Heart disease in marathon runners: a review.
AU Noakes T D
SO Medicine and science in sports and exercise, (1987 Jun) 19 (3) 187-94.
Ref: 46
Journal code: 8005433. ISSN: 0195-9131.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)
LA English
FS Priority Journals; Space Life Sciences
EM 198708
ED Entered STN: 19900305
Last Updated on STN: 19900305
Entered Medline: 19870807

L4 ANSWER 12 OF 13 MEDLINE on STN
AB Support for the concept that neurohormonal mechanisms play an important role in determining the survival of patients with severe chronic heart failure is derived from two lines of evidence: circulating levels of neurohormones are markedly elevated in patients who have a poor long-term prognosis and the survival of high-risk patients may be favorably modified by treatment with specific neurohormonal antagonists. Plasma norepinephrine is a major prognostic factor in patients with severe chronic heart failure, the most markedly elevated levels being observed in patients with the most unfavorable long-term prognosis. Data from uncontrolled studies suggest that low-dose beta-blockade may improve the survival of patients with dilated **cardiomyopathy**. Similar trends were noted in the Beta-Blocker **Heart Attack** Trial, in which patients with congestive heart failure before or accompanying their acute myocardial infarction experienced a significant reduction in sudden death when treated with beta-blockers. In contrast, there appeared to be little selective benefit in patients without heart failure, who presumably had low circulating levels of catecholamines. Similarly, serum sodium concentration is a major prognostic factor in patients with severe chronic heart failure, the shortest survival being observed in patients with the most severe hyponatremia. The poor long-term outcome of hyponatremic patients appears to be related to the marked elevation of plasma renin activity in these individuals, since (in retrospective studies) hyponatremic patients appeared to fare significantly better when treated with converting-enzyme inhibitors than when treated with vasodilator drugs that did not interfere with angiotensin II formation. In contrast, there appeared to be no selective benefit of converting-enzyme inhibition on the survival of patients with a normal serum sodium concentration, in whom plasma renin activity was low. These data suggest that neurohormonal systems may exert a deleterious effect on the survival of some patients with severe chronic heart failure, which may be favorably modified by long-term treatment with specific neurohormonal antagonists.
AN 87188170 MEDLINE
DN PubMed ID: 2882867
TI Role of neurohormonal mechanisms in determining survival in patients with severe chronic heart failure.
AU Packer M; Lee W H; Kessler P D; Gottlieb S S; Bernstein J L; Kukin M L
NC K04-HL-01229 (NHLBI)
R01-HL-25055 (NHLBI)
T32-HL-07347 (NHLBI)
SO Circulation, (1987 May) 75 (5 Pt 2) IV80-92. Ref: 110
Journal code: 0147763. ISSN: 0009-7322.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198706
ED Entered STN: 19900303
Last Updated on STN: 19970203
Entered Medline: 19870601

L4 ANSWER 13 OF 13 CA COPYRIGHT 2004 ACS on STN
AB Studies were performed in anesthetized controls dogs and in dogs with acute low-output heart failure produced by inflation of a balloon in the

thoracic inferior vena cava to determine the effects of synthetic atrial natriuretic peptide (ANF 8-33 [93590-01-3]) on renal function and renin [9015-94-5] release in this acute high-renin, Na-retaining preparation Intrarenal infusion of ANF 8-33 (0.3 μg/kg/min) resulted in decreases in arterial pressure and renal blood flow in both groups. Glomerular filtration rate increased in both low-output (Δ +10.7 mL/min) and control (Δ +8.7 mL/min) groups. Fractional Li excretion, a marker of proximal tubule reabsorption, also increased in both low-output (Δ +12.0%) and control (Δ +14.3%) groups. Renin secretory rate decreased in the low-output group from 852.8 to 149.5 ng/min and in the control group from 308.5 to 44.5 ng/mL. Intrarenal infusion of ANF 8-33 resulted in an attenuated increase in both urinary Na excretion (Δ +42.3 vs. Δ +201.2 μequiv/min) and fractional excretion of Na (Δ +0.48% vs. Δ +2.85%) in the low-output as compared with the control group. Thus, the administration of ANF 8-33 results in an increase in glomerular filtration rate and a decrease in proximal tubule reabsorption, as estimated by Li excretion, in both control dogs and those with acute low-output heart failure. Furthermore, despite a decrease in arterial pressure, synthetic ANF 8-33 markedly inhibits renin secretion under control conditions and in the high-renin state. Despite similar increases in glomerular filtration rate and decreases in proximal tubule reabsorption and renin release, the natriuretic response to ANF 8-33, although present, is markedly attenuated in this preparation of acute exptl. heart failure.

AN 103:190556 CA
TI Effects of synthetic atrial natriuretic peptide on renal function and renin release in acute experimental heart failure
AU Scriven, Terry A.; Burnett, John C., Jr.
CS Dep. Med., Mayo Med. Sch., Rochester, MN, 55905, USA
SO Circulation (1985), 72(4), 892-7
CODEN: CIRCAZ; ISSN: 0009-7322
DT Journal
LA English